

Rupture of a papillary muscle of the tricuspid valve

Echocardiographic diagnosis

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SUMMARY Abnormal tricuspid valve structure and motion resulting from the rupture of a right ventricular papillary muscle were visualised by two dimensional echocardiography. These findings were confirmed at operation. Two dimensional echocardiography appears to be a satisfactory method for evaluating patients with tricuspid regurgitation of sudden onset with a view to surgery.

Rupture of the right ventricular papillary muscle is relatively rare.¹ Because the haemodynamic consequences of the resulting severe tricuspid regurgitation can often be tolerated for fairly extended periods there should be ample time for diagnosis and surgical repair of this abnormality. Cardiac catheterisation and angiography provide important information, but details of the valve are usually obscured by the simultaneous dense opacification of atrium and ventricle. We describe the visualisation of this abnormality by two dimensional echocardiography.

Case report

A 25-year-old man was admitted for postoperative cardiac assessment. He had undergone heart surgery seven years previously for correction of Fallot's tetralogy. The tricuspid valve, which was regurgitant after damage by infective endocarditis, was also surgically repaired and he remained well until six months before this admission, when he noted progressive abdominal discomfort, palpitation, and pulsating neck veins. He had signs of congestive cardiac failure and severe tricuspid regurgitation and was in intermittent atrial flutter.

Significant physical findings on admission included a conspicuously high venous pressure with a prominent systolic wave, liver distension, ankle swelling, and evidence of ascites. The blood pressure was 110/60 mmHg and he was in sinus rhythm. There was cardiomegaly with a hyperdynamic apical impulse in the fifth intercostal space internal to the anterior axillary line. There was moderate left parasternal heave,

and a systolic thrill was palpable over the lower left sternal border. Cardiac auscultation disclosed evidence of residual right ventricular outflow tract obstruction and mild pulmonary regurgitation. In addition, there was a loud harsh pansystolic murmur best heard at the lower left sternal border radiating to the ventricular apex accentuating with inspiration, and an easily audible protodiastolic gallop.

The electrocardiogram documented sinus rhythm, first degree heart block, and right bundle-branch block, conduction abnormalities which had been present since surgery. There was pronounced cardiomegaly and small bilateral pleural effusions on the chest radiograph. Routine haematology and biochemistry studies were normal with the exception of the liver function tests. Blood cultures were negative.

The M-mode echocardiogram demonstrated normal left ventricular function and normal aortic and mitral valve movement and continuity. There was paradoxical septal motion and a very dilated right ventricle consistent with right ventricular volume overload. In addition, there were diastolic vibrations in the anterior leaflet of the tricuspid valve.

The two dimensional echocardiographic study using a HP77020A phased array imaging system showed similar findings, and in addition it showed frank flailing of the anterior tricuspid valve leaflet, prolapsing into an aneurysmally dilated right atrium during systole (Fig. 1 and 2). The inferior vena cava was very dilated and pulsatile; the pulmonary valve was inadequately visualised.

At cardiac catheterisation the pulmonary artery pressure was 30/8 mmHg and right ventricular pres-

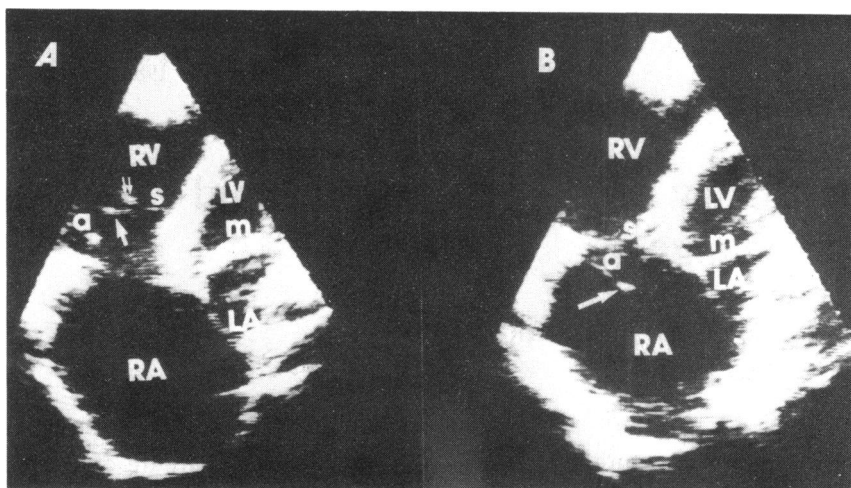


Fig. 1 Two dimensional echocardiogram: apical four chamber view. (A) Diastolic frame: the anterior (a) tricuspid leaflet (large arrow) has a thickened tip (small arrows) and overshoots the septal (s) leaflet. (B) Systolic frame: the anterior leaflet (a) (large arrow) has a flailing systolic motion and lies within the right atrium (RA) with a loss of systolic leaflet coaptation. LA, left atrium; m, mitral valve; RV, right ventricle; LV, left ventricle.

sure was 75/16 mmHg. The right atrial pressure was 20 mmHg with a "v" wave of 25 mmHg. There was no evidence of mitral regurgitation or a residual ventricular septal defect on left ventricular angiography. Right ventriculography showed considerable enlargement of the right ventricle, valvar and infundibular narrowing, and severe tricuspid regurgitation into the large right atrium. Pulmonary angiography was normal.

SURGERY

The right atrium was opened using a longitudinal incision. There was massive hypertrophy of the right ventricle with a small pulmonary artery and an aneurysmal right atrium. The tricuspid valve was severely regurgitant because of complete detachment of the head of the papillary muscle attaching the anterior cusp which was prolapsing into the right atrium. There was no evidence of recent infective endocarditis. The tricuspid valve was replaced by a porcine prosthetic valve and the valvar and infundibular obstruction relieved. The postoperative course was uncomplicated.

Discussion

Rupture of the right ventricular papillary muscle is rare, and occurs as a complication of right sided infective endocarditis² after myocardial infarction,³ penetrating and non-penetrating chest trauma,⁴ external cardiac massage,¹ and in association with primary pulmonary hypertension.⁵ The increased wall tension of the dilated right ventricle resulting from residual outflow tract obstruction and an abnormal tricuspid valve probably contributed to spontaneous papillary muscle rupture in this patient. Though catheterisa-

tion and cineangiography permit the assessment of the haemodynamic state and the severity of tricuspid regurgitation, the exact underlying anatomical changes cannot usually be fully determined. Standard M-mode echocardiography and two dimensional echocardiography have been of value in the non-invasive detection of many disorders of the tricuspid valve.⁶ The M-mode study permitted documentation of the right ventricular volume overload and graphically displayed the diastolic oscillations of the tricuspid valve. The latter, though non-specific,⁶ suggests disruption of leaflet integrity. The echoes recorded by this method, however, are usually limited to the anterior tricuspid leaflet in systole and the rapid opening motions in early diastole. Specific spatial anatomical information was provided by the wide sector arc of two dimensional echocardiography which visualised all three tricuspid leaflets (Fig. 1 and 2). This technique permitted visualisation of non-specific features of tricuspid regurgitation such as excessive systolic pulsation of the inferior vena cava and abnormal motion of the interatrial septum. More importantly, it characterised the exact nature of the disordered valve anatomy with a normal posterior leaflet, restricted motion of the septal leaflet (caused by previous surgery), and the flail nature of the anterior leaflet.

Right ventricular papillary muscle rupture has been documented by two dimensional echocardiography only once before in a patient with active endocarditis.⁷ A mobile mass of echoes attached to the tip of the chordae and the presence of tricuspid valve prolapse are very suggestive of ruptured right ventricular papillary muscle.

It may not always be possible to determine whether rupture is at muscle or at chordal level; the clinical and surgical implications of ruptured chordae, however, are similar.

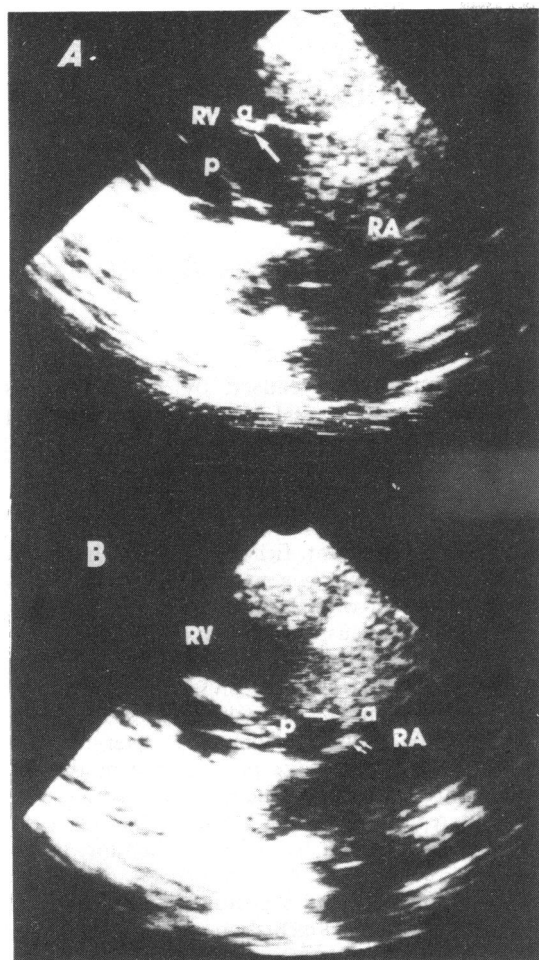


Fig. 2 Parasternal long axis view: (A) diastolic (B) systolic frames, visualising the right atrium (RA) and inflow region of the right ventricle (RV), showing prolapse of the anterior leaflet (a) (large arrow) with a thickened tip (small arrows) suggestive of papillary muscle rupture. p, posterior tricuspid valve leaflet.

A complete profile of the disordered valve structure can be obtained by two dimensional echocardiography and this appears to be a satisfactory non-invasive method for evaluating certain patients with tricuspid regurgitation of sudden onset with a view to surgical intervention.

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